

Case 2

Anne D. was an Indian girl of 16 years, the wife of the first patient described. The history and physical findings were similar to those of her husband. She was approximately five months pregnant and had apparently received no antepartum care. Urinalysis showed many leukocytes and culture of a mid-stream urine specimen produced a heavy growth of *Enterococcus*. Her ESR was 40 mm. and chest x-ray was normal. A biopsy of the gastrocnemius muscle confirmed the presence of larvae of *Trichinella spiralis*.

She was started on prednisone, 40 mg. per day, on the sixth hospital day, and discharged on a dosage of 7.5 mg. four times daily.

On March 19, 1971 she delivered a full-term normal male infant weighing 8½ lbs., which appeared perfectly healthy on careful examination. A year after her original infestation the mother appeared in good health, but a persistent leukocytosis of 11,000 per c.mm. was present, with 7% eosinophils. The cause of this is not known; no investigations for parasites were done at this time.

Discussion

The last reported outbreaks of trichinosis in Canada were due to pork.^{1, 2} In one outbreak thiabendazole was used in the treatment of 11 cases but the author concluded that it conferred no definite benefit.¹

The black bear has been recognized as a vector of trichinosis in the Arctic and cases in Alaska, Labrador and Northern Russia are reported.³⁻⁵ We are not aware of a previous report from the Prairie provinces.

The transplacental transmission of trichinosis with intrauterine infestation of the fetus has been reported in experimental animals, and in presumptive cases in humans.^{6, 7} The paucity of reports suggests that this must be a rare occurrence; despite the presumably heavy infection in Case 2, no abnormality was found in the child at delivery.

Résumé

Epidémie de trichinose en Saskatchewan, causée par consommation de viande d'ours avariée

Les auteurs rapportent une épidémie de trichinose dans une tribu d'Indiens Chippewayens vivant dans le Nord de la Saskatchewan et qui avaient mangé de la viande d'ours insuffisamment cuite. Le diagnostic a été basé sur l'anamnèse et sur l'examen clinique et confirmé par biopsie musculaire et par la découverte d'une forte éosinophilie. Les

sept malades ont guéri. Les deux malades qui ont été étudiés de façon plus approfondie ont été traités uniquement par la prednisone et ont guéri sans incidents: un an plus tard, on ne notait aucune séquelle. Une femme qui était enceinte de cinq mois au moment de l'infection a donné le jour à un enfant à terme et parfaitement normal.

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Trichinosis presenting as acute myocardial infarction

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Summary: A 20-year-old male patient is presented as a case of trichinous myocarditis with clinical symptoms and electrocardiographic evidence of an acute inferior myocardial infarction. He recovered rapidly and completely without any specific therapy. This seems to be a distinct rarity, having never been previously reported, but is of importance because of the almost uniformly excellent prognosis in this condition in contradistinction to that of a bona fide myocardial infarction occurring at this age.

Although in 1936 Cushing¹ stated that "cases of trichinosis with clinical and ECG evidence of myocardial damage are rare", more recent reviews have shown that among patients with clinically apparent trichinosis, myocardial involvement as witnessed by ECG abnormalities occurs in between 21 and 23%.^{2, 3} These abnormalities include conduction defects, non-specific T-wave flattening or inversion, and arrhythmias.^{1, 2, 4-6} However, there has never been reported a case of trichinous myocarditis presenting

with focal ischemic changes resembling an acute myocardial infarction. Such a case was seen at the Royal Victoria Hospital in July 1968.

Case report

A 20-year-old Sicilian-born male restaurant kitchen helper was admitted because of severe retrosternal pain of several hours' duration. Family history was entirely negative and the patient had never been ill prior to this episode. He had not left Canada since his arrival two years previously.

Two days before admission the patient noted low grade fever, malaise, and vague soreness in his shoulders and thighs. On the day of admission he experienced three episodes of severe dull retrosternal pain without radiation, each lasting 10 minutes, and all occurring within a few hours. He did not vomit, nor did he experience dyspnea, orthopnea, or other discomfort. He had not had a sore throat, joint pains, urinary discoloration or other symptoms.

Physical examination revealed a healthy looking young man in moderate distress due to chest pain as described above. Oral temperature was 100° F., pulse was 88 and regular, blood pressure was 120/80. He had 1+ periorbital edema but no edema elsewhere. Findings on examination of the chest were normal. Auscultation of the heart revealed an atrial gallop without other abnormalities. There was slight deltoid tenderness but no neurological or any other abnormality.

An electrocardiogram done in the emergency room showed insignificant

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q waves in leads II, III, and avF, ST elevation in these leads and in V-6, and reciprocal ST depression in leads V-1-V-2 (Fig. 1). This was interpreted as probable acute inferior wall myocardial infarction and he was admitted to the cardiac monitor unit. Laboratory tests revealed a hemoglobin of 14.7 g., hematocrit 49%, and leukocyte count 12,300 with 6% eosinophilia. ESR was 4 mm. in the first hour. BUN, serum creatinine, electrolytes, uric acid, cholesterol, two hour p.c. blood sugar, PBI, serum electrophoresis, LE preparations, anti-nuclear antibodies, rheumatoid factor, antistreptolysin-o titer serum complement and Paul Bunnell test were all within normal limits. Cultures from the throat and of stool and urine for virus were negative, as were all routine bacterial cultures. SGOT was 46 units per 100 ml. on admission, and fell on the second day to 16, remaining normal thereafter; LDH was normal throughout. Chest x-ray was negative.

Serial ECGs revealed a very rapid sequence of changes. A second tracing done six hours after admission showed decreasing ST elevation in II, III, and avF, and new T wave inversion in lead III. Twelve hours later the T wave was inverted in II, III, and avF (Fig. 2). By the next day this inversion was less, and within three days the ST and T wave changes were minimal and found only in lead III (Fig. 3). Over the next few weeks ECGs continued to demonstrate variable changes indicative of inferior wall ischemia. There was never any conduction defect or arrhythmia.

The eosinophilia noted on admission increased to a maximum of 22% on the tenth hospital day and was 13% by the third week of hospitalization. Because of

the absence of enzyme, leukocyte count and ESR changes, the unusual sequence of ECG changes, the eosinophilia, and the periorbital edema, a trichina skin test and trichinella agglutination test were done, both of which were weakly positive. The agglutination test repeated three weeks later was strongly positive, indicating a four-fold increase in the level of antibody. At this point the patient admitted to eating raw pork at least once weekly when the meat arrived at the restaurant in which he worked. X-rays of soft tissues were normal, as was an EEG. Muscle biopsy done two weeks after admission showed widespread myositis with eosinophilic and polymorphonuclear infiltration, but no larvae were detected.

The patient was treated by watchful waiting. His chest pain did not recur in hospital and fever, which varied between 101° and 102° during the first four days, disappeared thereafter. There was never evidence of cardiac failure, and the atrial gallop heard on admission soon disappeared leaving no abnormalities detectable by physical examination. When the diagnosis of trichinous myocarditis was considered firm, the patient was given a three-day course of thiabendazole which produced no change in his then asymptomatic course. When seen in clinic several months later, he had had no return of his symptoms and the ECG was normal. He had given up eating raw pork.

Comment

Despite the many cases of myocardial involvement in trichinosis reported in the literature, not one example could be found with ST segment elevation and the ECG signs of focal myocardial ischemia exhibited by our patient. That he had trichinosis and not another form of myocarditis seems sure. Although larvae were not found in the muscle biopsy, it should be remembered that biopsy performed earlier than the third week in the course of the disease may be unrewarding in this respect, and that even in the absence of demonstrable larvae, focal areas of acute necrosis with eosinophilic

and polymorphonuclear infiltrations with or without associated angitis are consistent with the diagnosis.^{2, 7} The four-fold increase in antibody titre as shown by serum trichinella agglutination is of great diagnostic significance.⁸ Of interest is the low ESR throughout this patient's course which has been reported in trichinosis.⁹

It is of paramount importance to distinguish this disease from a true myocardial infarction because of the great difference in prognosis between the two conditions. That a *bona fide* myocardial infarction occurring at age 20 is of grave prognostic importance is well known; the outcome of trichinous myocarditis is quite different. In reviewing 1023 patients, Gray found that although 23% of those with trichinosis had myocardial involvement, only one fatality could be attributed to the myocarditis.² That this serious complication of trichinosis is usually reversible is explained by its pathology. Zenker,¹⁰ in 1860, was the first to demonstrate larvae in the myocardium of a patient who died of trichinosis myocarditis. The condition produces an active cellular proliferation, with localized areas of necrosis and hemorrhage scattered throughout the heart muscle.¹¹ These changes are thought to be due to the occurrence and migration of larvae in the myocardium. However, no encystment has ever been found in heart muscle, despite both clinical and experimental studies on the subject.^{12, 13}

Because the larvae disappear from the myocardium, usually within two weeks, and no permanent encystment ever takes place therein, even the most serious cases of trichinous myocarditis will remit, providing proper treatment is given during the acute phase of the disease. Treatment, when there is evidence of serious cardiac compromise, consists of ACTH or corticosteroid therapy in high doses,^{9, 14} along with careful monitoring of cardiac function and treatment of failure if need arises.

Because the treatment and prognosis of trichinous myocarditis differ from these aspects of acute myocardial infarction, we feel this relatively rare situation is worthy of serious consideration in any youth who presents with symptoms and ECG evidence of acute myocardial infarction, especially if any of the other stigmata of trichinosis are

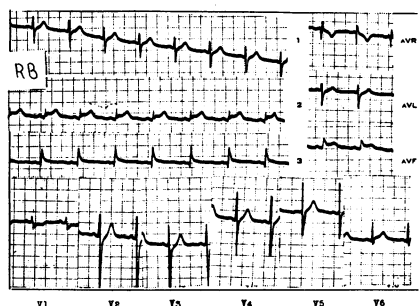


FIG. 1—Electrocardiogram taken at the time of admission to the hospital.

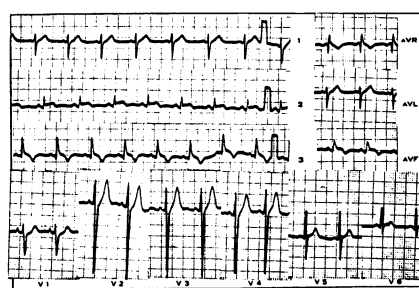


FIG. 2—Electrocardiogram taken 18 hours after admission.

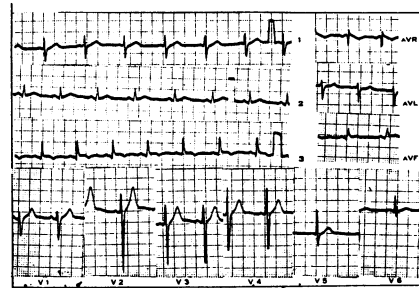


FIG. 3—Electrocardiogram taken on the fourth hospital day.

for
depression
& associated
anxiety*

Elavil

(amitriptyline hydrochloride, MSD Std.)

INDICATIONS. ELAVIL* (amitriptyline hydrochloride, MSD Std.) is recommended in the treatment of mental depression. ELAVIL* is also recommended in nocturnal enuresis — commonly a manifestation of an emotional problem. In cases where organic pathology has been excluded, ELAVIL* has been found effective in reducing the incidence of bed-wetting in some children.

DOSAGE. *Initial dose:* 25 mg. three times a day. If it is necessary to increase the dose, it is preferable to increase it by increments of 25 mg. and to add these doses in the evening. It is seldom necessary to exceed a total daily dose of 150 mg.

Maintenance: The most common maintenance dose is 25 mg. two to four times a day.

Elderly patients and adolescents: Ten mg. three times a day with 20 mg. at bedtime may be satisfactory.

Outpatients: It is seldom necessary to use doses in excess of 150 mg. a day in the management of depression in the patient who can be treated effectively as an outpatient.

Hospitalized patients: Most patients respond to doses of no more than 150 mg. a day. A small percentage of patients may require doses of up to 300 mg. a day.

Enuresis: A dosage of 10 mg. at bedtime has been found effective in children under six years of age. Doses of up to 25 mg. are required by some patients.

PRECAUTIONS. Because of its anticholinergic activity, ELAVIL* is contraindicated in patients with glaucoma and in patients who may be expected to experience problems of urinary retention. Also because the clinical experience and follow-up in pregnancy have been limited, ELAVIL* is not recommended for use in pregnant patients at this time. The side effects which may occur with ELAVIL* include drowsiness, dizziness, nausea, excitement, hypotension, fine tremor, jitteriness, weakness, headache, heartburn, anorexia, increased perspiration, incoordination, numbness, tingling of the limbs, possible peripheral neuropathy, and tachycardia, blurred vision, constipation, urinary retention, dryness of the mouth, monilial infection and other oral pathology secondary to dryness of the mouth have been reported to be associated with the use of amitriptyline hydrochloride in combination with other medication having anticholinergic activity or alone. Rarely, allergic type reactions have occurred, manifested by skin rash, or swelling of the face and tongue and itching. Agranulocytosis and jaundice have rarely been reported to have occurred in patients receiving amitriptyline hydrochloride. Although the etiologic role of the drug is uncertain, careful observation of all patients is recommended. High doses may cause temporary confusion or disturbed concentration.

Paralytic ileus has been reported very rarely, and only when other possible contributing factors have been involved. Nevertheless especially in the elderly, both physician and patient should be alert to this possibility, and take appropriate measures if constipation develops.

When patients who have been receiving a monoamine oxidase inhibitor are to be treated with ELAVIL*, it is recommended that at least two weeks be allowed to elapse between administration of the two agents to permit recovery from the effects of the monoamine oxidase inhibitor.

Combined use with other antidepressants may result in potentiation. Activation of latent schizophrenia and epileptiform seizures in chronic schizophrenics has been reported; the possibility of provoking mania or hypomania in manic-depressive patients should be borne in mind. Reversible ECG changes including flattening or inversion of T waves and bundle branch block have been reported in elderly patients.

It should be also borne in mind that the possibility of suicide in seriously depressed patients is inherent in the illness and may remain until significant remission occurs.

Patients receiving this drug should be cautioned against driving a car or operating machinery requiring alert attention. As with any psychotherapeutic agent, patients should be cautioned against errors of judgment due to change in mood and modification of the response to alcohol.

Detailed information is available to physicians, on request.

HOW SUPPLIED. Ca 3286 — Injection ELAVIL*, 10 mg./cc., is a clear, colorless solution, and is supplied in 10 cc. vials. Ca 3287 — Tablets ELAVIL*, 10 mg., are blue, biconvex, discoid-shaped film coated tablets, 1/4 of an inch in diameter, and are supplied in bottles of 100 and 500.

Ca 3288 — Tablets ELAVIL*, 25 mg., are yellow, biconvex, discoid-shaped film coated tablets, 1/4 of an inch in diameter, and are supplied in bottles of 100 and 500.

Ca 3301 — Syrup ELAVIL* (amitriptyline pamoate, MSD Std.), is a light red syrupy suspension containing in each 5 ml. amitriptyline pamoate equivalent to 10 mg. amitriptyline and is supplied in bottles of 225 cc. and 2250 cc.

Ca 8655 — Tablets ELAVIL*, 50 mg., are beige, biconvex, discoid-shaped film coated tablets, 1/4 of an inch in diameter, and are supplied in bottles of 50 and 1,000.

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present, such as periorbital edema, muscle aching or eosinophilia, or if there is a history of ingestion of raw pork.

Résumé

Cas de trichinose se présentant sous la forme d'un infarctus aigu du myocarde

Un homme de 20 ans se présente comme un cas de myocardite trichineuse, dont les symptômes cliniques et les signes de l'ECG évoquent un infarctus aigu du myocarde. Le malade a guéri rapidement et complètement sans recours à un traitement spécifique quelconque. D'après nous, ce cas est une entité clinique rare qui n'a jamais été rapportée. Il a cependant une réelle importance, étant donné l'excellent pronostic de cette pathologie, par opposition au cas classique de l'infarctus du myocarde qui survient à cet âge.

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